CORRESPONDENCE

Human dignity must be basis for debate on primate research

SIR — Bill Crum emphasizes a fundamental keynote of biomedical-research ethics in his Correspondence 'It should be possible to replace animals in research' (*Nature* **457**, 657; 2009) by stating that "good medical science" is not necessarily "morally justifiable or morally acceptable". On the other hand, many states and societies claim 'freedom of research' — meaning research being free from the need for justification — as a basic right. On the face of it, this looks like a discrepancy.

However, we have to recognize the fact that this freedom, like every other kind of freedom, has its ethical limits. Research can only be a right as long as it is not acting against our fundamental moral value: respect for human dignity. This is the basic point that we should agree on, regardless of our different opinions on what might constitute a breach of that principle.

With human dignity in mind, the ethical discussion about research on non-human primates has to focus on answering two questions. First, would prohibiting studies on primates constitute a threat to the human dignity of future generations, by reducing their chances of what we could consider a good life, as Roberto Caminiti states in his Correspondence 'Replacement of animals in research will never be possible' (*Nature* **457**, 147; 2009)? Second, is performing "invasive medical experiments" on creatures that "provide excellent experimental models of human cognition", as Crum states, a threat to our own dignity and our vision of how a good life should be led?

Only by using human dignity as the normative correlate for ethical decisions can we ensure that these decisions will be made on a basis that is equally important to all parties in this debate.

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Readers are welcome to comment at http://tinyurl.com/c62pgf

Rare BSE mutation raises concerns over risks to public health

SIR — Atypical forms (known as H- and L-type) of bovine spongiform encephalopathy (BSE) have recently appeared in several European countries as well as in Japan, Canada and the United States. This raises the unwelcome possibility that variant Creutzfeldt-Jakob disease (vCJD) could increase in the human population.

Of the atypical BSE cases tested so far, a mutation in the prion protein gene (*PRNP*) has been detected in just one, a cow in Alabama with BSE; her healthy calf also carried the mutation (J. A. Richt and S. M. Hall *PLoS Pathog.* **4**, e1000156; 2008). This raises the possibility that the disease could occasionally be genetic in origin. Indeed, the report of the UK BSE Inquiry in 2000 suggested that the UK epidemic had most likely originated from such a mutation and argued against the scrapie-related assumption.

Such rare potential pathogenic *PRNP* mutations could occur in countries at present considered to be free of BSE, such as Australia and New Zealand. So it is important to maintain strict surveillance for BSE in cattle, with rigorous enforcement of the ruminant feed ban (many countries still feed ruminant proteins to pigs). Removal of specified risk material, such as brain and spinal cord, from cattle at slaughter prevents infected material from entering the human food chain.

Routine genetic screening of cattle for *PRNP* mutations, which is now available, could provide additional data on the risk to the public. Because the point mutation identified in the Alabama animals is identical to that responsible for the commonest type of familial (genetic) CJD in humans, it is possible that the resulting infective prion protein might cross the bovine–human species barrier more easily. Patients with vCJD continue to be identified. The fact that this is happening less often should not lead to relaxation of the controls necessary to prevent future outbreaks.

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Scientific links with Cuba flourished despite US embargo

SIR — In your Editorial 'Cuba's biotech boom' (*Nature* **457**, 130; 2009), you state that "despite many constraints on interaction between Cuban and US scientists, biotech has prospered". In fact, US biotechnologists contributed in no small way to its development.

At the start, during the early 1980s, Cuban biotechnology was confined to a small house in a Havana suburb. An American group organized by Harlyn Halvorson, then director of Brandeis University's Rosenstiel Center and an inspirational leader, stepped in to help the venture. We were received warmly in Cuba whenever we visited.

The biotechnology effort soon transferred to a larger house across the street and from 1986 was housed in the majestic Center for Genetic Engineering and Biotechnology. The Cuban scientists set up symposia where one or more of us would speak.

The US government allowed us to travel to Cuba on the condition that we spent no American dollars there. We therefore continued to advise this fledgling group until the Soviet Union ceased to support Cuba financially and they could no longer pay for our visits.

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Idea of a love drug was no mystery to Shakespeare

SIR — In his Essay 'Love: neuroscience reveals all' (*Nature* **457**, 148; 2009), Larry Young claims that the biochemical understanding of love is not poetry. But at least one poet, namely William Shakespeare, foretold the application of drugs to manipulate the brain systems associated with pair bonding.

In *A Midsummer Night's Dream*, Oberon maintains that topical applications of the juice of the wild pansy (*Viola tricolor*, called 'love-in-idleness' in the play) "Will make or man or woman madly dote Upon the next live creature that it sees" (Act 2, Scene 1). The potion proves highly effective, supplying much of the humour in the play as Titania falls in love with the donkey-headed Bottom. Shakespeare also suggests that other substances from "Dian's bud" — variously identified as a species of wormwood (*Artemisia* spp.) or chaste tree (*Vitex agnus-castus*, a species not native to England but long known for its anti-libidinal properties) — could reverse the neurobiological results of the pansy. Perhaps poets have something to teach us about neurobiology and love after all.

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研究報告の概要

○プリオン病最新情報　英国:国立CJDサーベイランスユニット、月次vCJD・CJD統計、2009年1月5日時点

英国のCJDサーベイランスユニットから公表されたvCJDを始めとするプリオン病の患者数に関する最新情報である。2008年は、12月31日時点で140名の照会があった。内訳は、孤発性CJDによる死亡患者:73名、医原性CJDによる死亡患者:5名、GSS:3名、家族性CJD:2名、vCJD:1名。vCJD確定例または可能性例総数は前月から変化なく167名のままである。このデータは英国におけるvCJD流行は減少しつつあるとする見解に一致する。死亡患者数のピークは2000年の28名であり、その後2001年に20名、2002年に17名、2003年に18名、2004年に9名、2005年に5名、2006年に5名、2007年に5名、2008年に1名と減少している。

報告企業の意見	今後の対応
英国CJDサーベイランスユニットの統計によると、2009年1月5日の時点で、vCJD死亡患者総数には前月から変化なく167名のままであり、英国におけるvCJD流行は減少しつつあるとする見解に一致するとの報告である。	日本赤十字社は、vCJDの血液を介する感染防止の目的から、献血時に過去の海外渡航歴(旅行及び居住)を確認し、欧州36ヶ国に一定期間滞在したドナーを無期限に献血延期としている。また、英国滞在歴を有するvCJD患者が国内で発生したことから、平成17年6月1日より1980～96年に1日以上の英国滞在歴のある人の献血を制限している。今後もCJD等プリオン病に関する新たな知見及び情報の収集に努める。

使用上の注意記載状況・その他参考事項等
赤血球濃厚液-LR「日赤」 照射赤血球濃厚液-LR「日赤」 血液を介するウイルス、細菌、原虫等の感染 vCJD等の伝播のリスク

(19)

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Archive Number 20090108.0076
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PRION DISEASE UPDATE 2009 (01)
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A ProMED-mail post
<http://www.promedmail.org>
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International Society for Infectious Diseases
<http://www.isid.org>

[With the continuing decline in the number of cases in the human population of variant Creutzfeldt-Jakob disease -- abbreviated previously as vCJD or CJD (new var.) in ProMED-mail -- it has been decided to broaden the scope of the occasional ProMED-mail updates to include other prion-related diseases. Data on vCJD cases and other forms of CJD: sporadic, iatrogenic, familial, and GSS (Gerstmann-Straussler-Scheinker disease) are included also when they have some relevance to the incidence and etiology of vCJD. - Mod.CP

In this update:
[1] UK: National CJD Surveillance Unit - monthly statistics as of 5 Jan 2009
[2] France: Institut de Veille Sanitaire - as of 30 Dec 2008
[3] US National Prion Disease Pathology Surveillance Center - as of 30 Nov 20
[4] and [5] Prion protein function
[6] CJD Update

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[1] UK: National CJD Surveillance Unit - monthly statistics as of 5 Jan 2009
Date: Mon 5 Jan 2009
Source: UK National CJD Surveillance Unit, monthly statistics [edited]
<http://www.cjd.ed.ac.uk/figures.htm>

The number of suspect cases of vCJD referred to the CJD surveillance unit in Edinburgh and the number of deaths of definite and probable variant Creutzfeldt-Jakob disease [abbreviated in ProMED-mail as CJD (new var.) or vCJD], the form of the disease thought to be linked to BSE (bovine spongiform encephalopathy), remain unchanged since the previous monthly report; that is, the number of definite or probable vCJD cases (dead and alive) remains 16

This situation is consistent with the view that the vCJD outbreak in the UK is in decline. The 1st cases were observed in 1995, and the peak number of deaths was 28 in the year 2000, followed by 20 in 2001, 17 in 2002, 18 in 2003, 9 in 2004, 5 in 2005, 5 in 2006, 5 in 2007, and only one so far (up to the end of 2008).

Totals for all types of CJD cases in the year 2008

As of 31 Dec 2008 in the UK, so far there have been 140 referrals, 73 deaths from sporadic CJD, 5 deaths from iatrogenic CJD, 3 from GSS, 2 from familial CJD, and one from vCJD.

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[2] France: Institut de Veille Sanitaire - as of 30 Dec 2008
Date: 30 Dec 2008
Source: IVS - Maladie de Creutzfeldt-Jakob et maladies apparentees [French, trans. & summ. Mod.CP, edited]
<http://www.invs.sante.fr/display/?doc=publications/mcj/donnees_mcj.html>

During the period 1992 to 2008, there were 23 cases of vCJD, all now deceased. They occurred between 1996 and 2007: one case in 1996, one in 2000, one in 2001, 3 in 2002, none in 2003, 2 in 2004, 6 in 2005, 6 in 2006, 3 in 2007, and none so far in 2008. There were 12 male and 11 female patients.

Their ages at time of death ranged from 19 to 58 years (mean 39); 6 of the patients resided in the Ile-de-France [Paris area] and 17 in the provinces. All the cases were met-met homozygotes for codon 129 of the prion protein gene. No special risk factors were evident, which distinguished these patients from those with other forms of CJD (sporadic, genetic, iatrogenic). However, one patient had visited the UK at regular intervals.

Totals for all types of CJD cases in the year 2008

As of 30 Dec 2008 in France, during the course of 2008 there have been 1438 referrals, 76 deaths from sporadic CJD, 3 deaths from iatrogenic CJD, 8 from familial CJD, none from GSS, and none from vCJD.

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[3] US National Prion Disease Pathology Surveillance Center - as of 30 Nov 2
Date: 30 Nov 2008
Source: US National Prion Disease Pathology Surveillance Center [edited]
<http://www.cjdsurveillance.com/#esources-casereport.html>

Cases examined - as of 30 Nov 2008

During the period 1997 to 30 Nov 2008, 2 cases of vCJD were reported, both contracted overseas. The 1st case was recorded in 2004, disease contracted in the UK, and the 2nd in 2006, disease contracted in Saudi Arabia.

Totals for all types of CJD cases in the year 2008 as of 30 Nov 2008

So far in 2008 there have been 332 referrals, 199 cases of prion disease, including 151 cases of sporadic CJD, 21 cases of familial CJD, no cases of atrogenic CJD and no indigenous cases of vCJD.

Overall during the period 1997 to 2008, there have been 3018 referrals, 1745 cases of prion disease, 1456 cases of sporadic CJD, 252 cases of familial CJD, 4 cases of iatrogenic CJD and no indigenous cases of vCJD.

[During 2008 so far the USA with approximately 2.5x the combine populations of the UK and France have reported a similar number of cases of sporadic CJD (149 versus 151). Whether this is due ot a difference in surveillance procedure or actual disease incidence is unclear at the present time. - Mod.CP]

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[4] Prion protein function
Date: Sun 21 Dec 2008
Source: BBC News online [edited]
<http://news.bbc.co.uk/1/hi/health/7788444.stm>

Scientists sniff out prion secret

The brain protein which has a hand, when defective, in the lethal disease CJD may also be involved in aiding our sense of smell. Mice bred to lack the prion protein could not find buried food or choose between smells. Columbia University scientists said some symptoms of prion disease might be due to the loss of the protein's original role. The study was published in the journal Nature Neuroscience [see below].

The prion protein has historically received something of a bad press, being blamed in its misshapen form for degenerative brain diseases in humans and other animals. However, many scientists have been trying to uncover what it actually does when it is behaving correctly. Dr Stuart Firestein's team believe that one of these roles is to help us smell. While his prion-protein free mice were still able to detect scents, they had lost some higher functions which required that smell information to be analysed and processed by the brain. The scientists found changes in the communication between neurons in the nerve cells of the olfactory bulb, part of the forebrain which deals with odours. When the protein was restored to this part of the brain, the ability to discriminate between odours came back.

The scientists said that while the discovery had no direct link to the diseases caused by faulty prion proteins, it might help account for some of the symptoms experienced by patients, which might be due to the failure of the proteins to do their normal job properly, rather than the damage caused by accumulation of defective prions.

This is not the 1st suggested role for the prion protein -- in 2007, Leeds University scientist Professor Nigel Hooper said that it might help reduce the formation of "plaques" linked to the onset of Alzheimer Disease. He said of the newly-reported research: "It's likely that these proteins have a number of roles in various different body systems, including the olfactory system, as suggested here. "I don't think you can say that it is so mysterious any more, or that we do not understand what it does."

[Reference: Nature Neuroscience, Published online: 21 December 2008 doi:10.1038/nn.2238
<http://www.nature.com/neuro/journal/v12/n1/abs/nn.2238.html>

Title: Olfactory behavior and physiology are disrupted in prion protein knockout mice
Authors: Claire E Le Pichon1, Matthew T Valley1, Magdalini Polymenidou2,3, Alexander T Chesler1, Botir T Sagdullaev1,3, Adriano Aguzzi2 & Stuart Firestein1

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Abstract: The prion protein PrPC is infamous for its role in disease, but its normal physiological function remains unknown. Here we found a previously unknown behavioral phenotype of Prnp-/- mice in an odor-guided task. This phenotype was manifest in three Prnp knockout lines on different genetic backgrounds, which provides strong evidence that the phenotype is caused by a lack of PrPC rather than by other genetic factors. Prnp-/- mice also showed altered behavior in a 2nd olfactory task, suggesting that the phenotype is olfactory specific. Furthermore, PrPC deficiency affected oscillatory activity in the deep layers of the main olfactory bulb, as well as dendrodendritic synaptic transmission between olfactory bulb granule and mitral cells. Notably, both the behavioral and electrophysiological alterations found in Prnp-/- mice were rescued by transgenic neuronal-specific expression of PrPC. These data suggest that PrPC is important in the normal processing of sensory information by the olfactory system.]

[And from the same issue of Nature Neuroscience. See below - Mod.CP]

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[5] Prion protein function
Date: Sun 21 Dec 2008
Source: Nature Neuroscience 12, 7 - 8 (2009) [edited]
<http://www.nature.com/neuro/journal/v12/n1/full/nn0109-7.htm>

Title: Sniffing out a function for prion proteins

Abstract

When prion proteins go wrong, they can do serious damage, but little is known about their normal function, despite their ubiquitous expression in the brain. A new report in this issue [see above] suggests a critical role for prions in olfactory discrimination.

Introduction

Although the word prion was coined by Stanley Prusiner to describe the "proteinaceous infectious particle" that causes a family of fatal neurodegenerative diseases known as transmissible spongiform encephalopathies more than 20 years ago, little is known about the normal function of prion proteins. Most of what is known about them comes from studies of their involvement in these devastating diseases, which include Creutzfeld-Jakob disease, bovine spongiform encephalopathy ('mad-cow disease') and chronic wasting disease in elk and deer. These diseases are distinguished by rapidly progressive neurological deterioration and a pattern of neurodegeneration that is characterized by prominent vacuolization of neuronal cytoplasm, which gives the brain a sponge-like histological appearance. The key pathogenic event in these diseases is the conversion of an endogenous cell-surface glycoprotein, the prion protein (PrPc), to a pathological isoform (PrPsc) that has an abnormal conformation and an unusual resistance to proteolytic degradation. PrPsc accumulates in cells and plaque-like extracellular deposits, converting more PrPc into the pathogenic form and triggering neurodegeneration by mechanisms that are still not fully understood. Conversion of PrPc can be a result of inherited mutations, infection of the

...ase with a prion-infected tissue or rare sporadic events. Although the formation of PrPsc is believed to result in a gain of toxic function, a loss of function of PrPc has not been excluded as being involved in prion disease PrPc is most abundantly expressed in the brain and it would be expected that the loss of this protein would result in substantial neurobehavioral modifications. However, the specific role of PrPc in neural function and behavior is far from clear. In fact, previous work suggests that the most robust phenotype of PrPc loss in transgenic mice is protection from prion diseases. Although changes in PrPc expression influence a variety of critical cellular processes in neurons, including cell survival, synaptic maintenance and plasticity, and axonal maintenance, data on these issues have occasionally been contradictory. Thus, 'elusive' remains one of the descriptors most commonly attached to this protein in papers and reviews on PrPc. Fortunately, a clue to the elusive prion function may lie right under, in, our noses. Le Pichon and colleagues have begun this investigation in this issue [see proceeding report].

There are several major hurdles to learning about the function of a particular protein. One of these is knowing where the protein resides in cells. This localization can help narrow down the potential functions of the protein. Earlier this year [2008], it was demonstrated, using new highly specific antibodies, that PrPc in the olfactory system is localized to the axons of both peripheral olfactory sensory receptor neurons and central neurons such as the mitral cells of the olfactory bulb. Glia or support cells in the olfactory bulb or olfactory epithelium were not detectably labeled. In addition to axons, PrPc was also observed in the dendritic spines of axonless olfactory bulb granule cells. These spines are both pre- and postsynaptic to mitral cells, forming reciprocal synapses. Combined with the axon staining, this suggests a potential role for PrPc in presynaptic function. However, given how widely expressed PrPc is throughout the brain, simply showing its presence in the olfactory system was only circumstantial; further tests were required to determine whether it has a functional role in olfaction.

The observation that PrPc is expressed in olfactory sensory neurons, mitral cells and granule cells raises the possibility that it is important for the local circuit function of the olfactory bulb. Olfactory sensory neurons in the nose send axons directly into the brain, terminating on mitral cells, which send their axons directly to olfactory cortex. In the olfactory bulb, local circuits, which include granule cells, refine spatiotemporal patterns of sensory neuron input, and this local circuit function can be monitored electrophysiologically through oscillations in local field potentials. Previous work in a variety of laboratories has demonstrated that manipulation of local circuit function in the olfactory bulb can modulate various aspects of odor perception, 7). Thus, the stage was set to ask whether loss of PrPc affects normal olfaction. Le Pichon and colleagues provide a convincing affirmative answer and with it a clue to PrPc function. Specifically, the loss of PrPc in neurons of the olfactory system of transgenic mice impairs odor-guided behaviors such as finding buried food and simple odor discrimination. The deficit was expressed

...anosmia but was rather an apparent impairment in odor discrimination per se. Although PrPc is found in olfactory sensory neurons, the behavioral deficits were not associated with detectable changes in receptor function. In fact, the sense of smell could be rescued by selectively replacing PrPc in olfactory bulb neurons alone, suggesting a central brain site of action.

Given that PrPc deletion disrupted odor-guided behavior, the final question is raised of whether or not there are neural correlates of this behavioral change in the olfactory bulb. Using electrophysiological recordings, Le Pichon et al. demonstrated specific changes in local circuit function in the olfactory bulb in the PrPc knockouts. For example, using in vivo electrical stimulation to assay local circuit interneuron function, the authors found a decrease in inhibition of mitral cells by granule cell interneurons. This mitral cellgranule cell reciprocal interaction has been hypothesized to be important for everything from lateral inhibition to odor memory to state-dependent modulation of olfactory bulb function. Physiologically, activity in this local feedback circuit underlies high-frequency oscillations in olfactory bulb activity in response to odor stimulation. These olfactory bulb local field potential oscillations may facilitate temporal coding and/or binding of disparate odor features by target neurons in the olfactory cortex. Le Pichon et al. found that these odor-evoked high-frequency oscillations were abnormal in PrPc knockout mice.

The results suggest that PrPc may be important in local circuit function in the olfactory system and may in turn influence odor perception. There has been some debate over whether neural damage done by prion diseases is solely caused by the buildup of PrPsc or whether the concomitant loss of PrPc may also be involved. By demonstrating a systems-level effect of PrPc loss, Le Pichon et al. suggest that both may be important.

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2 Ralph A Nixon is at the Center for Dementia Research, Nathan Kline Institute for Psychiatric Research, 140 Old Orangeburg Road, Orangeburg, New York 10962, USA, and the Departments of Psychiatry and Cell Biology, New York University School of Medicine, 550 1st Ave, New York, New York 10016, USA. <dwilson@nki.rfmh.org>]

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[The references cited in the text can be found by accessing the original text of tis report in Naute Neuroscinece using the URL at the beginining of the report. - Mod.CP]

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[6] CJD Update
Date 12 Dec 2008
Source: Health Protection Agency Report, Emerging Infections/CJD [abbreviated and edited]

<http://www.hpa.org.uk/hpr/infections/ei_cjd>.htm>

Creutzfeldt-Jakob disease (CJD) update report

This 6-monthly report provides an update on reports of incidents of potential iatrogenic (healthcare-acquired) exposure to CJD via surgery, and on the National Anonymous Tonsil Archive. Data are correct as of 5 Dec 2008. For numbers of CJD case reports, readers should consult data provided by the national CJD Surveillance Unit (NCJDSU), Edinburgh [1], and the ProMED-mail monthly Prion Disease Updates]. The latest yearly analysis of vCJD reports (onsets and deaths) is also available from the NCJDSU Web site [2], and the ProMED-mail monthly Prion Disease Update.

Reports of incidents of potential iatrogenic exposure to CJD via surgery: 1 Jan 2000 to 30 Jun 2008

There were a total of 350 incidents reported during this period (tabulated in the original text). 12 surgical incidents were reported between 1 Jan and 30 Jun 2008. A surgical incident occurs when a patient undergoes surgery but is only identified as having CJD or being at risk of CJD at a later date. (This means that the ACDP TSE Working Group infection control guidelines would not have been followed). The surgery carried out on an index patient with, or at risk of CJD, may result in contamination of the instruments with abnormal prion protein. (A table in the original text gives the number of CJD surgical incidents reported to the CJD Incidents Panel from January 2000 to June 2008 by the diagnosis of the index patient.)

Investigation of surgical incidents may result in advice to remove surgical instruments from clinical use (to quarantine, destroy, or donate for research). Such advice is generally only given for instruments considered to be potentially contaminated with the CJD agent that have not undergone a certain number of cycles of use and decontamination since their use on an index patient. Hospitals are asked to consider sending any instruments to be permanently removed from use to the Surgical Instrument Store (held by the Health Protection Agency, Porton Down) for research. In the 2nd half of 2007, there were no incidents in which instruments were permanently removed from use.

The Panel may advise contacting and informing some patients of their possible exposure to CJD in a surgical incident. Such advice is generally only given for patients who have definitely been exposed to potentially contaminated instruments which have been used on risk tissues in certain index patients. The Panel may advise that some of these patients should be considered "at-risk of CJD for public health purposes" and asked to take certain precautions (i.e., not to donate blood or other tissues and to inform their medical and dental carers prior to any invasive procedures) in order to reduce the risk of transmitting the CJD agent further. Since 2000, 20 incidents have given rise to such advice (tabulated in the original text). One of these incidents was reported in the 1st half of 2008. The Panel has so far categorised 64 patients as "at-risk"; 13 of whom died before notification. 3 patients have not been notified due to local, clinical decisions. (One index patient undergoing a cataract operation was also a blood component

[illegible] with evidence of vCJD infection.]

National anonymous tonsil archive for studies of detectable abnormal prion protein
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The National Anonymous Tonsil Archive (NATA) continues to receive approximately 400 tonsil pairs per week. The archive had received a total of 67 696 tonsil pairs up to the end of October 2008 from hospitals in England and Scotland. A further 3000 tonsil pairs have been received from the Medical Research Council Prion Unit. Therefore the total number of tonsil pairs in the archive was 70 696.

Testing of homogenates of the tonsil tissue from the archive began at the end of January 2007. 2 enzyme immunoassays (EIAs) are being used for the initial screening of the homogenates for the presence of abnormal prion protein. These EIAs allow the identification of any tonsils that need to be investigated further by the more specific tests of Western blotting (WB) and immunohistochemistry (IHC) [4].

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[1] The National Creutzfeldt-Jakob Disease Surveillance Unit, The University of Edinburgh. CJD statistics. CJD figures. Edinburgh: NCJDSU, 3 May 2005. Available at <http://www.cjd.ed.ac.uk/figures.htm>.

[2] The National Creutzfeldt-Jakob Disease Surveillance Unit, The University of Edinburgh. Incidence of variant Creutzfeldt-Jakob Disease Onsets and Deaths in the UK January 1994 - March 2005.Edinburgh: NCJDSU, 14 Apr 2005. Available at <http://www.cjd.ed.ac.uk/vcjdqdec06.htm>.

[3] HPA CJD Incidents Panel [online]. London: HPA. Available at <http://www.hpa.org.uk/web/HPAweb&Page&HPAwebAutoListName/Page/1204031511121>

[4] Spongiform Encephalopathy Advisory Committee. Combining evidence from tissue surveys to estimate the prevalence of subclinical vCJD. SEAC, 2008. Available at <http://www.seac.gov.uk/papers/paper100-2.pdf>.

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[see also:
2008

Prion disease update 2008 (14): new vCJD wave imminent? 20081218.3980
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